BIOGRAPHICAL SKETCH

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NAME: Rehermann, Barbara

eRA COMMONS USER NAME (credential, e.g., agency login): REHERMANN

POSITION TITLE: Chief, Immunology Section, Liver Diseases Branch, NIDDK, NIH

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Medizinische Hochschule, Hannover, Germany	M.D.	1991	Medicine
The Scripps Research Institute, La Jolla, CA	Postdoctoral	1993-1995	Immunology

A. Personal Statement

I am a senior investigator at the National Institutes of Diabetes and Digestive and Kidney Diseases at the NIH, Bethesda. For more than 20 years I have conducted translational studies on antiviral immunity using biospecimens from virus-infected patients, animal models and in vitro models of virus-host cell interaction. In particular, I have studied (i) the role of innate and adaptive immune responses in the clearance and pathogenesis of hepatitis virus and liver disease pathogenesis, (ii) mechanisms of chronic immune activation in persistent infection, (iv) the effects of long-term exposure to inflammatory cytokines and viral antigen on innate immune cells, NK cells and T cells, and their reversibility by the antiviral therapy. I bring both clinical expertise and immunology research expertise to this interdisciplinary research.

Relevant to the present application, we have in recent years examined the regulation of intrahepatic and systemic immune responses by the gut microbiome. We have created mouse models to evaluate beneficial health effects of natural microbiota on antiviral immunity and disease resistance, and demonstrated that these effects are mediated by regulation of inflammation.

B. Positions and Honors

1991-1992 Medical Intern, Internal Medicine, University Clinic, Essen, Germany
1992-1993 Medical Resident, Department of Gastroenterology and Hepatology, Medizinische Hochschule, Hannover, Germany
1993-1995 Postdoctoral Fellow, Department of Exp Medicine, The Scripps Research Institute, La Jolla, CA
1995-1998 Gastroenterology/Hepatology Fellow and Principal Investigator, Viral and Autoimmune Hepatitis Research, Dep. Gastroenterology and Hepatology, Medizinische Hochschule, Hannover, Germany
1998-2004 Tenure-Track Investigator, Liver Diseases Section, NIDDK, NIH
2005- Chief, Immunology Section, Liver Diseases Branch, NIDDK, NIH

Honors: DAAD Scholar (German Academic Exchange Service), Bonn, Germany (1988-1989); Scholarship of Cusanuswerk, Bonn, Germany (1988-1991); Research Award for Best thesis in Exp. Research, Medizinische Hochschule, Hannover, Germany (1995); Young Investigator Award, 4th European Gastroenterology Week, Berlin, Germany (1995); Dr. Norbert Henning Award, University of Erlangen-Nürnberg, Germany (1996); Young Investigator Award, European Association for the Study of Liver Diseases, London (1997); Pettenkofer Research Award, Max von Pettenkofer Foundation, Munich, Germany (1997); Bench-to-Bedside Award, National Institutes of Health, Bethesda, MD, USA (2001,2002); 17th Annual Stephen L. Winter Memorial Hepatology Lectureship, U of Illinois at Chicago (2001); Research Excellence in Gastrointestinal and Liver Disease (REGAL) Award, U Kansas (2001); elected member, American Society for Clinical Investigation

(2004); Loeffler-Frosch Award of the German Society for Virology (2005) Salzman Award Scientific Committee (2007-2010), Japan Society for Promotion of Science Fellowship Committee (2008-2010), HHMI-NIH Research Scholars Program Advisory Committee (2008-2011); NIH Immunology Interest Group Steering Committee (2009-2010); AASLD Basic Research Committee (2009-2011); NIDDK Nancy Nossal Scientific Mentorship Award (2010); NIH Merit Award (2013); elected member, Association of American Physicians (2014); Norman P. Salzman Memorial Mentor Award in Virology (2015)

Professional Societies: AAI, AAP, AASLD, ASCI (councilor 2008-2011, secretary/treasurer 2009-2011), ASM

Editorial Boards: Journal of Hepatology (2000-present); Hepatology (2002-1006, 2008-2011, 2017-present); Gastroenterology (2002-2007, 2016-present); Journal Virology (2005-2013; 2016-present), Journal of Infectious Diseases (2006-present)

Associate Editor: Journal of Immunology (2006-2010); ***Consulting Editor:*** The Journal of Clinical Investigation (2008-present); ***Academic Editor:*** Plos Medicine (2007, 2008)

Ad Hoc Reviewer: Gastroenterology, Genes and Immunity, Gut, Hepatology, Immunity, J. Clin. Invest., J. Exp. Med., J. Gen. Virol., J. Hepatology, J. Immunology, J. Infect. Diseases, J. Interferon Cytokine Research, J. Virology, Nature, Nature Immunology, Nature Medicine, Nature Reviews Immunology, New England Journal of Medicine, Plos Medicine, PNAS, Science, The Lancet, Trends in Microbiol., Trends in Mol. Med., Vaccine

C. Contribution to science (total of 141 publications)

1. Relevant to the present application, I have studied hepatitis B virus (HBV) infection for more than two decades. My early work defined the nature of protective T cell and antibody-based immunity from hepatitis B virus infection. Specifically, I have shown that antiviral T cells and antibodies control persistent low levels of transcriptionally active HBV for decades after recovery from acute viral hepatitis. More recently, research in my laboratory demonstrated that vaccine-induced immunity to HBV is protective but not sterilizing. This is important because HBV is not cleared but controlled by the host immune response and can re-activate when host immune responses are suppressed, e.g. by chemotherapy. Finally, my laboratory also characterized immune responses in chronic HBV infection.

    a. **B. Rehermann**, P. Fowler, J. Sidney, J. Person, A. Redeker, M. Brown, M. Moss, A. Sette, F. V. Chisari. The cytotoxic T lymphocyte response to multiple hepatitis B virus polymerase epitopes during and after acute viral hepatitis. The Journal of Experimental Medicine, 177:751-762, 1995
    b. **B. Rehermann**, C. Pasquinelli, S. M. Mosier, F. V. Chisari. Hepatitis B virus (HBV) sequence variation in CTL epitopes is not common in patients with chronic HBV infection. The Journal of Clinical Investigation, 961:527-1534, 1995
    c. **B. Rehermann**, C. Ferrari, C. Pasquinelli, F. V. Chisari. The hepatitis B virus persists for decades after patients' recovery from acute viral hepatitis despite active maintenance of a cytotoxic T lymphocyte response. Nature Medicine, 2:1104-1108, 1996
    d. A. Penna, M. Artini, A. Cavalli, M. Levrero, A. Bertoletti, M. Pilli, F. V. Chisari, **B. Rehermann**, G. Del Prete, F. Fiaccadori, C. Ferrari. Long-lasting memory T cell responses following self-limited acute hepatitis B. The Journal of Clinical Investigation, 98:1185-1194, 1996
    e. **B. Rehermann**, K. M. Chang, J. McHutchison, R. Kokka, M. Houghton, C. Rice, F. V. Chisari. Differential cytotoxic T lymphocyte responsiveness to the hepatitis B and hepatitis C viruses in chronically infected patients. Journal of Virology, 70:7092-7102, 1996
    f. **B. Rehermann**, D. Lau, J. H. Hoofnagle, F. V. Chisari. Cytotoxic T lymphocyte responsiveness after resolution of chronic hepatitis B virus infection. The Journal of Clinical Investigation, 97:1655-1665, 1996
    g. **B. Rehermann**. Intrahepatic T cells in hepatitis B: Viral control versus liver cell injury. The Journal of Experimental Medicine, 191:1263-1268, 2000
    h. **B. Rehermann**. Immune responses in hepatitis B virus infection. Seminars in Liver Diseases, 23:21-38, 2003
    i. T. Manigold, **B. Rehermann**. Chronic hepatitis B and hepatocarcinogenesis does prevention of collateral damage bring the cure? Hepatology, 37:707-710, 2003
    j. M. Nascimbeni, **B. Rehermann**. Determination of hepatitis B virus-specific CD8+ T-cell activity in the liver. Methods Molecular Medicine, 96:65-84, 2004
    k. E. Mizukoshi, J. Sidney, B. Livingston, M. Ghany, J. Hoofnagle, A. Sette, **B. Rehermann**. Cellular immune responses to the hepatitis B virus polymerase. The Journal of Immunology, 173:5863-5871, 2004

l. P. Vandepapelière, **B. Rehermann**, M. Koutsoukos, P. Moris, N. Garçon, M. Wettendorff, G. Leroux-Roels. Potent enhancement of cellular and humoral immune responses against recombinant hepatitis B antigens using AS02A adjuvant in healthy adults. Vaccine, 23:2591-2601, 2005
m. **B. Rehermann**, M. Nascimbeni. Immunology of hepatitis B and C virus infection. Nature Reviews Immunology, 5:215-229, 2005
n. V. Racanelli. **B. Rehermann.** The liver as an immunological organ. Hepatology, 43S54-62, 2006
o. **B. Rehermann**. Pathogenesis of chronic viral hepatitis: differential roles of T cells and NK cells. Nature Medicine 19:859-68114, 2013
p. J. M. Werner, A. Abdalla, N. Gara, M. G. Ghany, **B. Rehermann**. The hepatitis B vaccine protects re-exposed healthcare workers, but does not provide sterilizing immunity. Gastroenterology, 145:1026-1034, 2013
q. S. H. Park, **B. Rehermann**. Immune responses to HCV and other hepatitis viruses. Immunity, 40:13-24, 2014
r. **B. Rehermann**, A. Bertoletti. Immunological aspects of antiviral therapy of chronic hepatitis B virus and hepatitis C virus infections. Hepatology, 61:712-721, 2015
s. N. Gara, A. Abdalla, E. Rivera, X. Zhao, J. M. Werner, T. J. Liang, J. H. Hoofnagle, **B. Rehermann,** M. Ghany. Durability of antibody response against hepatitis B virus in healthcare workers vaccinated as adults. Clinical Infectious Diseases, 60: 505-513, 2015
t. **B. Rehermann**. Natural killer cells in viral hepatitis. Cellular and Molecular Gastroenterology Hepatology. 6:578-588, 2015
u. T. M. Block, S. Locarnini, B. J. McMahon, **B. Rehermann**, M. G. Peters. Use of current and new endpoints in the evaluation of experimental hepatitis B therapeutics. Clinical Infectious Diseases; 64:1283-1288, 2017
v. Bolte FJ, **Rehermann B**. Tissue-resident T cells in hepatitis B: A new target for cure? The Journal of Experimental Medicine 214:1564-1566, 2017
w. X. Cheng, Y. Xia, E. Serti, P. D. Block, M. Chung, **B. Rehermann,** K. Chayama, T. J. Liang. Hepatitis B virus evades innate immunity of hepatocytes but activates cytokine production by macrophages. Hepatology. Jun 30. doi: 10.1002/hep.29348. [Epub ahead of print], 2017

2. Relevant to the present application I have studied the regulation of intrahepatic and systemic immune responses by the gut microbiome. With a growing field of microbiome research, there has been a substantial re-appraisal of the gut-liver axis. In recent years, my laboratory conducted translational studies on the phenotype and function of innate and adaptive immune cells in all three compartments of the gut-liver axis (portal blood, systemic blood and liver) in patients with different stages of hepatitis virus-induced liver disease. We also studied the role of mucosal associated invariant T (MAIT) cells, which are enriched at barrier sites such as the gut and the liver and able to respond both inflammatory cytokines and to metabolites from E. coli and other riboflavin-synthesizing bacteria.

   Finally, we studied natural microbiota and demonstrated their host fitness-promoting traits in a new mouse model. This work received the 2017 NIH DDIR Innovation award. We showed that natural microbiota reduce systemic and local inflammation in disease models. This is illustrated by decreased levels of pro-inflammatory and increased levels of anti-inflammatory cytokines and reduced inflammatory cell infiltration along with higher survival rates during viral infection. It is further supported by less inflammation, reduced colitis-induced weight loss and tumor burden in a colorectal tumorigenesis model.

   a. F. J. Bolte, A. C. O'Keefe, L. M. Webb, E. Serti, E. Rivera, T. J. Liang, M. Ghany, **B. Rehermann**. Intra-hepatic depletion of mucosal associated invariant T cells in hepatitis C virus-induced liver inflammation. Gastroenterology, Aug 2. pii: S0016-5085(17)35975-9. doi: 10.1053/j.gastro.2017.07.043. [Epub ahead of print], 2017
   b. F. Bolte, A. C. O'Keefe, L. M. Webbn, O. Etzion, R. Ali, E. Levy, M. Ghany, T. Heller, **B. Rehermann**. Differential contribution of viral infection and bacterial translocation to liver inflammation in chronic viral hepatitis, submitted, 2017
   c. S. P. Rosshart, B. G. Vassallo, D. Angeletti, D. S. Hutchinson, A. P. Morgan, K. Takeda, H. D. Hickman, J. A. McCulloch, J. H. Badger, N. J. Ajami, G. Trinchieri, F. Pardo-Manuel de Villena, J. W. Yewdell, **B. Rehermann**. Wild mouse gut microbiota promotes host fitness and improves disease resistance. Cell, in press, 2017

3. Relevant to the present application, which includes genetic studies of virus-host interaction, I have studied the role of major histocompatibility complex (MHC) haplotypes, KIR-HLA compound genotypes and IFN-lambda SNPs in chronic viral hepatitis.

   a. E. Mizukoshi, M. Nascimbeni, J. B. Blaustein, K. Mihalik, C. M. Rice, T. J. Liang, S. M. Feinstone, **B. Rehermann**. Molecular and immunological significance of chimpanzee major histo-compatibility complex haplotypes for hepatitis C virus immune response and vaccination studies. Journal of Virology, 76:6093-6103, 2002

b. G. Ahlenstiel, M. P. Martin, X. Gao, M. Carrington, **B. Rehermann**. Distinct KIR/HLA compound genotypes affect the kinetics of antiviral NK cell responses. The Journal of Clinical Investigation, 118:1017-1026, 2008
c. L. Prokunina-Olsson, B. Muchmore, W. Tang, R. Pfeiffer, H. Park, H. Dickensheet, DHergott, P. Porter-Gill, A. Mumy, I. Kohaar, S. Chen, N. Brand, M. Tarway, L. Liu, F. Sheikh, J. Astemborski, H. Bonkovsky, B. Edlin, C. Howell, T. Morgan, D. Thomas, **B. Rehermann**, R. Donnelly, T. O'Brien. A variant upstream of IFNL3 (IL28B) creating a new interferon gene IFNL4 is associated with impaired clearance of HCV. Nat Genet 2013;45:164-71.
d. **B. Rehermann.** Peptide-dependent HLA-KIR-mediated regulation of NK cells. J Hepatology, 65: 237–239, 2016
e. H. Park, **B. Rehermann**. The role of genetics in liver disease progression. Hepatology (invited), 2017

4. Relevant to the current application, I have experience in studying human innate immune responses. My laboratory studied the role of interferons in viral hepatitis, and described a dichotomous NK cell function of increased cytotoxicity and decreased production of antiviral cytokines driven by virus-induced interferons.

a. E.C. Shin, U. Seifert, T. Kato, C. Rice, S. Feinstone, P. Kloetzel, **B. Rehermann**. Virus-induced type I IFN stimulates generation of immunoproteasomes at the site of infection. J Clin Invest, 116:3006-3014, 2006
b. E. C. Shin, U. Seifert, S. Urban, K.T. Truong, S. M. Feinstone, C.M. Rice, P. M. Kloetzel, **B. Rehermann.** Proteasome activator and antigen-processing aminopeptidases are regulated by virus-induced type I interferon in the hepatitis C virus-infected liver. Journal of Interferon and Cytokine Research, 27:985-990, 2007
c. G. Ahlenstiel, R. H. Titerence, C. Koh, B. Edlich, J. J. Feld, Y. Rotman, M. Ghany, J. H. Hoofnagle, T. J. Liang, T. Heller, **B. Rehermann**. Natural killer cell function is polarized towards cytotoxicity in chronic hepatitis C in an IFN-α dependent manner. Gastroenterology, 138:325-335, 2010
d. Ahlenstiel G, Edlich B, Hogdal LJ, Rotman Y, Noureddin M, Feld JJ, Holz LE, Titerence RH, Liang TJ, **Rehermann B**. Early changes in natural killer cell function indicate virologic response to interferon therapy for hepatitis C. Gastroenterology 2011;141:1231-9.
e. H. Park, E. Serti, O. Eke, B. Muchmore, L. Prokunina-Olsson, S. Capone, A. Folgori, **B. Rehermann**. IL-29 is the dominant type III interferon produced by hepatocytes during acute hepatitis C. Hepatology, 56:2060-2070, 2012
f. Prokunina-Olsson L, Muchmore B, Tang W, Pfeiffer RM, Park H, Dickensheets H, Hergott D, Porter-Gill P, Mumy A, Kohaar I, Chen S, Brand N, Tarway M, Liu L, Sheikh F, Astemborski J, Bonkovsky HL, Edlin B, Howell CD, Morgan TR, Thomas DL, **Rehermann B**, Donnelly RP, O'Brien TR. A variant upstream of IFNL3 (IL28B) creating a new interferon gene IFNL4 is associated with impaired clearance of HCV. Nat Genet 2013;45:164-71.
g. E. Serti, J. M. Werner, M. Chattergoon, A. L. Cox, V. Lohmann and **B. Rehermann**. Monocytes activate natural killer cells via inflammasome-induced IL-18 in response to hepatitis C virus replication. Gastroenterology,
h. **Rehermann B**. Natural killer cells in viral hepatitis. Cell Mol Gastroenterol Hepatol 2015;1:578-588.
i. F. J. Bolte, A. C. O'Keefe, L. M. Webb, E. Serti, E. Rivera, T. J. Liang, M. Ghany, **B. Rehermann**. Intra-hepatic depletion of mucosal associated invariant T cells in hepatitis C virus-induced liver inflammation. Gastroenterology, Aug 2. pii: S0016-5085(17)35975-9. doi: 10.1053/j.gastro.2017.07.043. [Epub ahead of print], 2017

5. We identified determinants of natural and vaccine-induced adaptive immunity to HCV and demonstrated that these are T cell-based.

a. A. Takaki, M. Wiese, G. Maertens, E. Depla, U. Seifert, A. Liebetrau, J. Miller, M. P. Manns, **B. Rehermann**. Cellular immune responses persist and humoral responses decrease two decades after recovery from a single-source outbreak of hepatitis C. Nature Medicine, 6:578-582, 2000
b. M. Nascimbeni, E. Mizukoshi, M. Bosmann, M. Major, K. Mihalik, C. M. Rice, S. M. Feinstone, **B. Rehermann**. Kinetics of $CD4^+$ and $CD8^+$ memory T cell responses during HCV rechallenge of previously recovered chimpanzees. Journal of Virology, 77: 4781-4793, 2003
c. U. Seifert, H. Liermann, M. Wiese, A. Halenius, T. Ruppert, H. Wedemeyer, K. Rispeter, P. Henklein, A. Sijts, H. Hengel, P. M. Kloetzel, **B. Rehermann**. HCV mutation in a single source outbreak affects proteasomal epitope processing. The Journal of Clinical Investigation, 114:250-259, 2004
d. T. Manigold, E.C. Shin, E. Mizukoshi, K. Mihalik, K. Murthy, C. Rice, C. Piccirillo, **B. Rehermann**. $Foxp3^+CD4^+$ T cells control virus-specific T cell memory in chimpanzees recovered from HCV. Blood 107:4424-4432, 2006
e. E. C. Shin, S. Capone, R. Cortese, S. Colloca, A. Nicosia, A. Folgori, **B. Rehermann**. The kinetics of hepatitis C virus-specific CD8 T cell responses in the blood mirror those in the liver in acute hepatitis C virus infection. Journal of Virology, 82:9782-9788, 2008
f. **Rehermann**. Hepatitis C virus versus innate and adaptive immune responses: a tale of co-evolution and coexistence. The Journal of Clinical Investigation, 119:1745-1754, 2009
g. E. Shin, S. Park, M. DeMino, M. Nascimbeni, K. Mihalik, M. Major, N. S. Veerapu, T. Heller, S. M. Feinstone, C. M. Rice, **B. Rehermann**. Delayed induction, not impaired recruitment of specific CD8 T cells causes the late onset of acute hepatitis C. Gastroenterology, 141:686-695, 2011

h. S. Park, E. Shin, S. Capone, L. Caggiari, V. De Re, A. Nicosia, A. Folgori, **B. Rehermann.** Successful vaccination induces multifunctional memory T-cell precursors associated with early control of hepatitis C virus. Gastroenterology, 143:1048-1060, 2012

i. E. C. Shin, S. Park, M. Nascimbeni, M. Major, L. Caggiari, V. de Re, S. Feinstone, C. Rice, **B. Reher-mann**. The frequency of CD127+ HCV-specific T cells but not the expression of exhaustion markers predict the outcome of acute hepatitis C virus infection. Journal of Virology, 87:4772-4777, 2013

6. We conducted a series of studies to assess the prevalence of protective T cell memory against HCV in humans. Specifically, we asked whether repeated exposure to low-dose antigen primes and maintains protective HCV-specific T cells in the absence of seroconversion. In a prospective study of health-care workers with documented low-level HCV exposure we demonstrated that innate (NKT and NK cells) and adaptive (HCV-specific CD4 and CD8 T cells) responses can indeed be induced in the absence of detectable viremia and seroconversion. We reproduced this immune response in an animal model by repeatedly exposing chimpanzees to human blood that contained trace amounts of HCV. However, the pre-existing T cell response declined rapidly and *de novo* T cell responses were not induced when the chimpanzees were challenged with HCV due to an increased frequency and an altered subset composition of regulatory T cells. The results suggest that repeated low-dose HCV exposure, e.g. in endemic areas and due to high-risk behavior, impedes the response to subsequent infection. They also imply that the efficacy of experimental HCV vaccines may be underestimated if these are tested in cohorts with prior exposure.

   a. E. Mizukoshi, C. Eisenbach, B.R. Edlin, K.P. Newton, C. Weiler-Normann, L.H. Tobler, M.P. Busch, M. Carrington, J.A. McKeating, T.R. O'Brien, **B. Rehermann**. Hepatitis C virus-specific immune responses of frequently exposed long-term injection drug users. The Journal of Infectious Diseases, 198:203-212, 2008

   b. J. M. Werner, T. Heller, A. M. Gordon, A. Sheets, A. H. Sherker, E. Kessler, K. S. Bean, M. Stevens, J. Schmitt, **B. Rehermann**. Innate immune responses in hepatitis C virus exposed healthcare workers who do not develop acute infection. Hepatology, 58:1621-1631, 2013

   c. T. Heller, J. Werner, F. Rahman, E. Mizukoshi, Y. Sobao, A. Gordon, A. Sheets, A. Sherker, E. Kessler, K. Bean, S. Herrine, J. Schmitt, **B. Rehermann**. Occupational exposure to HCV induces early T cell responses in the absence of seroconversion in a longitudinal cohort study. The Journal of Infectious Diseases, 208:1020-5, 2013

   d. S. Park, N. Veerapu, E. Shin, A. Biancotto, J. McCoy, S. Capone, A. Folgori, B. **Rehermann.** Subinfectious HCV exposures suppress T cell responses against subsequent acute infection. Nature Medicine, 19:1638-1642, 2013

   e. N. S. Veerapu, S. H. Park, D. C. Tully, T. M. Allen, **B. Rehermann**. Trace amounts of sporadically re-appearing HCV RNA can cause infection. The Journal of Clinical Investigation, 11924:3469-78, 2014

7. Finally, we used HCV treatment studies as a unique translational model to examine the reversibility of the phenotypic, molecular and epigenetic imprint that decades-long exposure to viral antigens and inflammatory cytokines leave on immune cells.

   a. J. Werner, E. Serti, X. Chepa-Lotrea, J. Stoltzfus, G. Ahlenstiel, M. Noureddin, J. Feld, T. Liang, Y. Rotman, B. **Rehermann**. Ribavirin improves the IFN-γ response of NK cells to IFN-based therapy of HCV. Hepatology 2014;60:1160-9.

   b. E. Serti, X. Chepa-Lotrea, Y. Kim, M. Keane, N. Fryzek, T. J. Liang, M. Ghany, **B. Rehermann**. Successful interferon-free therapy of chronic HCV infection normalizes NK cell function. Gastroenterology, 2015, 149: 190-200

   c. Serti E, Park H, Keane M, O'Keefe AC, Rivera E, Liang TJ, Ghany M, **Rehermann B**. Rapid decrease in hepatitis C viremia by direct acting antivirals improves the natural killer cell response to IFNalpha. Gut 2016.

D. Ongoing Research Support

1ZIA DK054508: Rehermann (PI) 09/01/1999-09/01/2021
Analysis and Modulation of Virus-Host Interaction in Infections of the Liver.

1ZIA DK054509: Rehermann (PI) 09/01/1999-09/01/2021
Immunology of Acute and Chronic Viral Hepatitis

1ZA DK054516: Rehermann (PI) 09/01/2008-09/01/2021
Immunology of Innate Immune Defenses in Viral Infections

NIH DDIR Innovation Award: Rehermann (PI) 2017
Creating animal models with natural microbiota to study disease resistance